CHAPTER 41

The Third and Fourth Heart Sounds

KEY TEACHING POINTS

- The third and fourth heart sounds (S_3 and S_4) both originate from rapid diastolic filling of one of the ventricles. They are collectively called *gallops*. The S_3 differs from the S_4 in timing and clinical significance.
- *Right ventricular gallops* appear at the left lower sternal border, intensify with inspiration, and are associated with abnormalities of the jugular venous waveforms. *Left ventricular gallops* appear at the apex and diminish in intensity during inspiration. All gallops are best heard with the bell of the stethoscope.
- The S_3 is an early diastolic sound. It is associated with a dilated ventricle, systolic dysfunction, and elevated filling pressures. The S_3 often quickly disappears after the patient is treated with diuretic medications.
- The S_4 is a presystolic sound. It is associated with a stiff ventricle, caused by ischemic, hypertensive, or hypertrophic cardiomyopathy. Once heard, the S_4 usually persists unless the patient develops atrial fibrillation. Unlike the S_3, the S_4 does not predict the patient's hemodynamic findings.

I. INTRODUCTION

Although the third and fourth heart sounds (S_3 and S_4) are both sounds that originate in the ventricle from rapid diastolic filling, they differ in timing and clinical significance. S_3 appears in early diastole and, if the patient is older than 40 years of age, the sound indicates severe systolic dysfunction or valvular regurgitation. In persons younger than 40 years of age, S_3 may be a normal finding (i.e., the *physiologic* S_3).[1] S_4 appears in late diastole, immediately before S_1, indicating that the patient's ventricle is abnormally stiff from hypertrophy or fibrosis. If discovered in persons of any age, the S_4 is an abnormal finding.

In the late 19th century the great French clinician Potain accurately described most features of S_3 and S_4, their pathogenesis, and their distinction from other double sounds, such as the split S_1 or split S_2.[2] In his writings he called them *gallops*, a term he attributed to his teacher Bouillard.[2,3]

II. DEFINITIONS

Several different terms have been used to describe these diastolic sounds.

A. GALLOP

A gallop is a triple rhythm with an extra sound in diastole (either S_3, S_4, or their summation). The term refers only to pathologic sounds (i.e., it excludes physiologic S_3) and, despite its connotation, a patient may have a gallop whether the heart rate is fast or slow.[2,4]

B. THIRD HEART SOUND (S_3)

The third heart sound is sometimes called the **ventricular gallop** or **protodiastolic gallop**.[2] It appears in early diastole, 120 to 180 ms after S_2.[5] To mimic the sound, the clinician should first establish the cadence of the normal S_1 (*lub*) and S_2 (*dup*):

lub dup lub dup lub dup

and then add an early diastolic sound (*bub*):*

lub du bub lub du bub lub du bub

The overall cadence of the S_3 gallop (*lub du bub*) is similar to the cadence of the word *Kentucky*.

C. FOURTH HEART SOUND (S_4)

The fourth heart sound is sometimes called the **atrial gallop** or **presystolic gallop**.[2] To mimic the sound, the clinician establishes the cadence of S_1 and S_2 (*lub dup*) and then adds a presystolic sound (*be*):

be lub dup be lub dup be lub dup

The cadence of the S_4 gallop (*be lub dup*) is similar to the cadence of *Tennessee*.†

D. SUMMATION GALLOP

The summation gallop is a loud gallop that occurs in patients with tachycardia. In fast heart rhythms, diastole shortens, causing the events that produce S_3 (rapid early diastolic filling) to coincide with those producing S_4 (atrial systole). The resulting sound sometimes is louder than the patient's S_1 or S_2.

Not all gallop rhythms in patients with tachycardia are summation gallops. The only way to confirm the finding is to observe the patient after the heart rate slows. (In the past, slowing was often induced by carotid artery massage, although in elderly patients this is no longer recommended. See Chapter 16.) If slowing causes the gallop to disappear or evolve into two distinct but fainter sounds (i.e., S_3 and S_4), it was a genuine summation gallop. If the sound evolves instead into a single S_3 or single S_4, it was not a summation gallop.[4,7]

E. QUADRUPLE RHYTHM

The quadruple rhythm consists of S_1, S_2, and both S_3 and S_4.[4] It is an uncommon finding, usually only evident in patients with slow heart rates. It is sometimes called the **train wheel rhythm** because the sound resembles that produced by the two pairs of wheels from adjacent train cars as they cross the coupling of a railroad track:[3,7]

be lub du bub be lub du bub be lub du bub

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* To pronounce the S_3 gallop with correct timing, the "p" of *dup* (S_2) must be dropped. In most patients the accent is on S_2 (*lub* ***du*** *bub*), although in others it falls on S_1 or S_3. The clinician can practice all three versions, always maintaining the same cadence, to become familiar with the varying sounds of S_3.

† Canadian teachers have suggested different mnemonics for the timing of S_3 and S_4: *Montreal* (pronounced MON TRE al) for S_3 and *Toronto* (tor ON to) for S_4.[6]

III. TECHNIQUE

A. LOCATION OF SOUND AND USE OF STETHOSCOPE

S_3 and S_4 are both low-frequency sounds (20 to 70 Hz), bordering on the threshold of hearing.[8] Therefore they are best heard with the bell of the stethoscope, applied lightly to the body wall with only enough force to create an air seal.[2,5] Gallops that originate in the left ventricle are best heard with the bell over the apical impulse or just medial to it. They are sometimes only audible with the patient lying in the left lateral decubitus position.[9] Gallops from the right ventricle are best heard with the bell over the left lower sternal border or, in patients with chronic lung disease, the subxiphoid area.[2,5]

B. RIGHT VERSUS LEFT VENTRICULAR GALLOPS

Aside from their different locations, other distinguishing features of right and left ventricular gallops are their response to respirations and association with other findings in the neck veins and precordium. Right ventricular gallops become louder during inspiration; left ventricular gallops become softer during inspiration.[10] The right ventricular S_4 may be associated with giant A waves in the neck veins and sometimes a loud presystolic jugular sound (see Chapter 36).[11] The left ventricular S_4 may be associated with a palpable presystolic movement of the apical impulse (see Chapter 38).

C. DISTINGUISHING THE S_4-S_1 SOUND FROM OTHER SOUNDS

Three combinations of heart sounds produce a double sound around S_1: (1) the S_4-S_1 sound, (2) split S_1, and (3) S_1-ejection sound. The following characteristics distinguish these sounds:[10]

1. USE OF THE BELL

The S_4 is a low-frequency sound, best heard with the bell. Firm pressure with the bell on the skin—which tends to remove low-frequency sounds—will cause the S_4-S_1 combination to evolve into a single sound, in contrast to the split S_1 and the S_1-ejection sound that remain double.

2. LOCATION

The S_4-S_1 sound is heard best at the apex, left lower sternal border, or subxiphoid area (see the section on Location of Sound and Use of Stethoscope). The split S_1 is loudest from the apex to lower sternal border but sometimes is also heard well over the upper left sternal area. The aortic ejection sound is heard from the apex to the upper right sternal border. The pulmonary ejection sound is restricted to the upper left sternal area.[12]

3. EFFECT OF RESPIRATION

Although the S_4 may become louder (RV S_4) or softer (LV S_4) during inspiration, respiration does not affect the interval between S_4 and S_1. In contrast, the split S_1 interval varies with respiration in up to one-third of patients.

Expiration makes the pulmonary ejection sound louder.[12] The aortic ejection sound does not vary with respiration.[13]

4. PALPATION

Only the S_4-S_1 sound is accompanied by a presystolic apical impulse (see Chapter 38). The intensity of the S_4 (i.e., by auscultation) correlates moderately with the

amplitude of the presystolic impulse on apexcardiography ($r = 0.46$, $p < 0.01$); similarly the palpability of the presystolic impulse correlates approximately with the amplitude of S_4 on phonocardiography ($r = 0.52$, $p < 0.01$).[14]

IV. PATHOGENESIS

A. NORMAL VENTRICULAR FILLING CURVES

Filling of the right and left ventricles during diastole is divided into three distinct phases (Fig. 41.1). The first phase, the rapid filling phase, begins immediately after opening of the atrioventricular valves. During this phase, blood stored in the atria rapidly empties into the ventricles. The second phase, the plateau phase (diastasis), begins at the moment the ventricles are unable to relax passively any further. Very little filling occurs during this phase. The third phase, atrial systole, begins with the atrial contraction, which expands the ventricle further just before the next S_1.

B. VENTRICULAR FILLING AND SOUND

Both S_3 and S_4 occur at those times during diastole when blood flow entering the ventricles temporarily stops (i.e., the S_3 appears at the end of the rapid filling phase, and the S_4 toward the peak of atrial systole) (Fig. 41.1). Sounds become audible if the blood *decelerates abruptly* enough, which transmits sufficient energy to the ventricular walls and causes them to vibrate (an analogy is the tensing of a handkerchief between two hands: abrupt tensing produces sound, whereas slow tensing is silent).[15-21] Two variables govern the suddenness of this deceleration and therefore whether gallops become audible: (1) the flow rate during entry and (2) stiffness of the ventricle. The greater the flow rate, the *louder* the sound. The stiffer the ventricle, the *higher the frequency* of the sound.[22] Because gallops consist of low frequencies that are difficult to hear (around 20 to 50 Hz), anything increasing their frequency content (i.e., stiff ventricles) makes the sound more likely to be heard.

Even though S_3 and S_4 both result from rapid flow rates into stiff ventricles, the diseases causing them differ completely.

C. THE THIRD HEART SOUND (S_3)

The S_3 gallop appears when early diastolic filling is exaggerated, which occurs in two types of cardiac disorders.

1. CONGESTIVE HEART FAILURE

The most common cause of the S_3 gallop is congestive heart failure from systolic dysfunction. In these patients the S_3 indicates that atrial pressure is abnormally elevated, an especially important finding in patients with dyspnea, implying that heart disease is the principal cause of the shortness of breath. In addition to elevated atrial pressure, these patients typically have a dilated cardiomyopathy and low cardiac output.[23,24] Although both high atrial pressure (causing rapid flow rates) and cardiomyopathy (causing stiff ventricles) contribute to the sound, atrial pressure is the more important clinical variable, because the sound disappears as soon as pressure falls after diuresis.

2. REGURGITATION AND SHUNTS

Patients with valvular regurgitation or left-to-right cardiac shunts also may develop an S_3 gallop, whether or not atrial pressure is high, because these

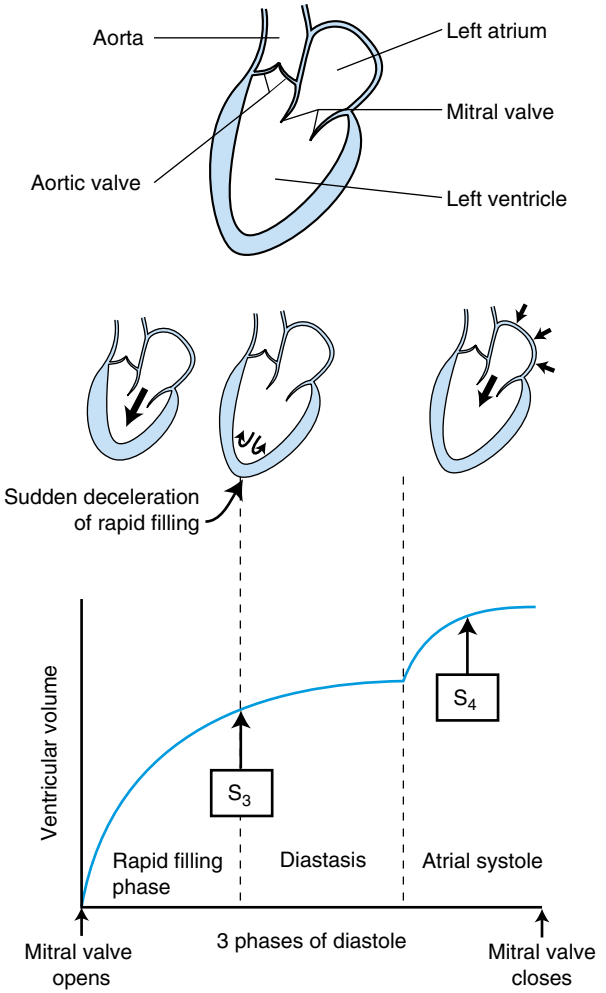

FIG. 41.1 TIMING OF THIRD AND FOURTH HEART SOUNDS. The figure depicts the three phases of diastolic filling of the left ventricle (*y*-axis on graph, ventricular volume; *x*-axis, time). The S_3 occurs at the end of the rapid filling phase, when passive filling suddenly decelerates. The S_4 occurs during atrial systole. Similar events on the right side of the heart may produce a right ventricular S_3 or S_4 (see text).

disorders all cause excess flow over the atrioventricular valves. Patients with mitral regurgitation, ventricular septal defect, or patent ductus arteriosus may develop a left ventricular S_3 from excess diastolic flow over the mitral valve into the left ventricle (in mitral regurgitation, the excess diastolic flow simply represents the diastolic return of the regurgitant flow). Patients with atrial septal defect may develop a right ventricular S_3 from excess flow over the tricuspid valve into the right ventricle.

D. THE FOURTH HEART SOUND (S_4)

The S_4 gallop occurs in patients with hypertension, ischemic cardiomyopathy, hypertrophic cardiomyopathy, or aortic stenosis—all disorders characterized by ventricles stiffened from hypertrophy or fibrosis.[2,23-25] Patients with the sound must be in sinus rhythm and have strong atrial contractions, and most have normal atrial pressures, normal cardiac output, and normal ventricular chamber size. Unlike the S_3, the S_4 is a durable finding that does not wax and wane unless the patient develops atrial fibrillation (and thus loses the atrial contraction).

E. SUMMATION GALLOP AND QUADRUPLE RHYTHM

The summation gallop occurs because fast heart rates shorten diastole, primarily by eliminating the plateau phase (Fig. 41.1), which brings the events causing S_3 close to those causing S_4. Diastolic filling is concentrated into a single moment, thus causing a very loud sound.

The quadruple rhythm typically occurs in patients who have had a long-standing S_4 gallop from ischemic or hypertensive heart disease but who then develop cardiac decompensation, high filling pressures, and an S_3.[7]

Rarely, an intermittent summation gallop may appear in patients with slow heart rates due to complete heart block (or VVI pacing).[26] The gallop appears only during those moments of atrioventricular dissociation when atrial systole and early diastole coincide (i.e., the P wave on the electrocardiogram falls just after the QRS). Although the sound is technically a summation gallop, the clinician perceives what sounds like an intermittent S_3.

F. PHYSIOLOGIC S_3

Persons younger than 40 years of age with normal hearts may also have an S_3 sound (i.e., physiologic S_3) because normal early filling can sometimes be so rapid that it ends abruptly and causes the ventricular walls to vibrate and produce sound. Compared with healthy persons lacking the sound, those with the physiologic S_3 are leaner and have more rapid early diastolic filling.[1] The physiologic S_3 disappears by age 40 because normal aging slows ventricular relaxation and shifts filling later in diastole, thus diminishing the rate of early diastolic filling and making the sound disappear.[27]

V. CLINICAL SIGNIFICANCE

A. THE THIRD HEART SOUND

1. CONGESTIVE HEART FAILURE

EBM Box 41.1 shows that the presence of the S_3 gallop is a significant finding indicating depressed ejection fraction (likelihood ratio [LR] = 3.4 to 4.1; see EBM Box 41.1), elevated left atrial pressures (LR = 3.9), and elevated B-type natriuretic peptide (BNP) levels (LR = 10.1). Other studies confirm its value as a predictor of poor systolic function.[35,44] The *absence* of the S_3 gallop argues that the patient's ejection fraction is greater than 30% (i.e., negative LR for ejection fraction <30% is 0.3; see EBM Box 41.1).

In patients with a history of congestive heart failure, the S_3 predicts responsiveness to digoxin[45] and overall mortality.[46]

2. VALVULAR HEART DISEASE

In patients with mitral regurgitation, the S_3 is a poor predictor of elevated filling pressure (LR not significant) and depressed ejection fraction (LR = 1.9).[47] Some studies correlate the sound with severity of mitral regurgitation,[20] whereas others do not.[47]

EBM BOX 41.1
*The Third and Fourth Heart Sounds**

Finding (Reference)	*Sensitivity (%)*	*Specificity (%)*	**Likelihood Ratio† if Finding Is**	
			Present	*Absent*
The Third Heart Sound				
Detecting ejection fraction <0.5[20,28-31]	11-51	85-98	**3.4**	0.7
Detecting ejection fraction <0.3[29,30]	68-78	80-88	**4.1**	**0.3**
Detecting elevated left heart filling pressures[31-34]	12-37	85-96	**3.9**	0.8
Detecting elevated BNP level[35,36]	41-65	93-97	**10.1**	0.5
Detecting myocardial infarction in patients with acute chest pain[37]	16	95	**3.2**	NS
Predicting postoperative pulmonary edema[38,39]	17	99	**14.6**	NS
Predicting postoperative myocardial infarction or cardiac death[38,39]	11	99	**8.0**	NS
The Fourth Heart Sound				
Predicting 5-year mortality in patients after myocardial infarction[40]	29	91	**3.2**	NS
Detecting elevated left heart filling pressures[33,41]	35-71	50-70	NS	NS
Detecting severe aortic stenosis[42,43]	29-50	57-63	NS	NS

*Diagnostic standard: for *ejection fraction*, left ventricular ejection fraction <0.5 or <0.3 (as indicated above) by scintigraphy or echocardiograph (see Chapter 48); for *elevated left heart filling pressures*, pulmonary capillary wedge pressure >12 mm Hg[32] or left ventricular end-diastolic pressure >15 mm Hg;[31,33,34,41] for *elevated BNP level*, ≥100 pg/mL[35] or >1525 pg/mL;[36] for *myocardial infarction*, development of new electrocardiographic Q waves, elevations of CK-MB, or both; for *severe aortic stenosis*, peak gradient >50 mm Hg[42] or valve area <0.75 cm^2.[43]
†Likelihood ratio (LR) if finding present = positive LR; LR if finding absent = negative LR.
NS, Not significant, *BNP,* B-type natriuretic peptide.
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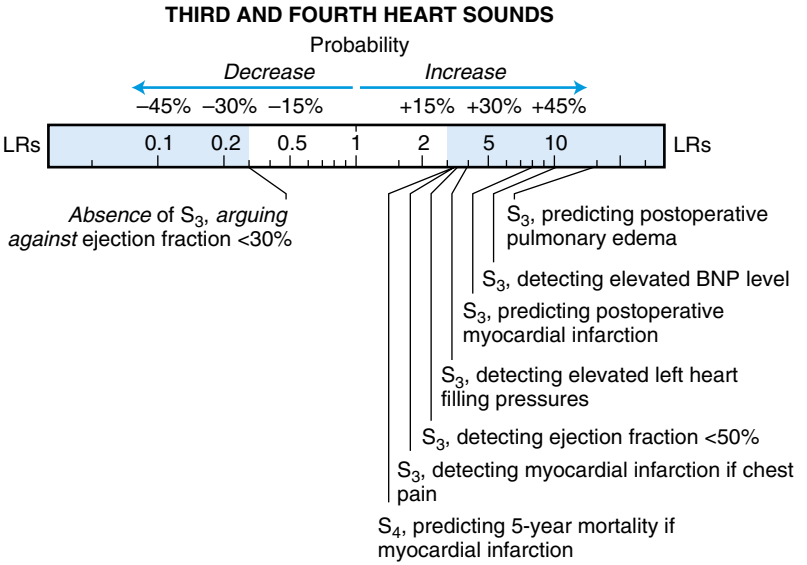

In contrast, the S_3 is a helpful finding in patients with aortic valve disease. In patients with aortic stenosis, the S_3 detects both elevated filling pressures (LR = 2.3 for pulmonary capillary wedge pressures ≥12 mm Hg) and depressed ejection fraction (LR = 5.7 for EF <50%).[47] In patients with aortic regurgitation the S_3 detects both severity of regurgitation (LR = 5.9 for regurgitant fraction ≥40%, see Chapter 45) and ejection fraction less than 50% (LR = 8.3).[20]

3. PATIENTS WITH ACUTE CHEST PAIN

In patients with acute chest pain presenting to emergency departments, the finding of an S_3 increases the probability of myocardial infarction (LR = 3.2; EBM Box 41.1).

4. PREOPERATIVE CONSULTATION

During preoperative consultation, the finding of S_3 is ominous, indicating that the patient, without any other intervention, has an increased risk of perioperative pulmonary edema (LR = 14.6) and myocardial infarction or cardiac death (LR = 8).[38]

B. THE FOURTH HEART SOUND

The finding of the S_4 gallop has less diagnostic value, simply because the disorders causing stiff ventricles are so diverse and because the S_4 does not predict the patient's hemodynamic findings. The finding does not predict ejection fraction, left heart filling pressures, or postoperative cardiac complications.[23,24,33,38,39] It also does not predict significant aortic stenosis in elderly patients with aortic flow murmurs, presumably because many patients with mild stenosis have the finding for other reasons, such as ischemic heart disease.[42,43]

Nonetheless, when detected 1 month after myocardial infarction, the S_4 is a modest predictor of 5-year cardiac mortality (LR = 3.2; see EBM Box 41.1). Experienced auscultators in the past did show that clinical deterioration in patients

with ischemic disease caused the S_4-S_1 interval to widen, which could be recognized at the bedside, but proper interpretation of this finding required knowledge of the patient's PR interval, thus limiting its utility.[48] In patients with chaotic heart rhythms, the finding of an S_4 excludes atrial fibrillation and suggests other diagnoses such as multifocal atrial tachycardia.

The S_4 is rare in patients with chronic mitral regurgitation, because the dilated atrium of these patients cannot contract strongly. Therefore finding an S_4 gallop in a patient with mitral regurgitation is an important clue to the diagnosis of *acute* mitral regurgitation (e.g., ruptured chorda tendineae; see Chapter 46).[49-51]

The references for this chapter can be found on www.expertconsult.com.

REFERENCES

1. Kupari M, Koskinen P, Virolainen J, Hekali P, Keto P. Prevalence and predictors of audible physiological third heart sound in a population sample aged 36 to 37 years. *Circulation*. 1994;89:1189–1195.
2. Craige E. Gallop rhythm. *Prog Cardiovasc Dis*. 1967;10(3):246–261.
3. Sloan AW. Cardiac gallop rhythm. *Medicine*. 1958;37:197–215.
4. Feinstein AR, Hochstein E, Luisada AA, et al. Glossary of cardiologic terms related to physical diagnosis and history. *Am J Cardiol*. 1968;21:273–274.
5. Ronan JA. Cardiac auscultation: the third and fourth heart sounds. *Heart Dis Stroke*. 1992;1:267–270.
6. Warnica JW. Canadian mnemonics for heart sounds. *Can Med Assoc J*. 2007;176(1):69.
7. Harvey WP, Stapleton J. Clinical aspects of gallop rhythm with particular reference to diastolic gallop. *Circulation*. 1958;18:1017–1024.
8. Tavel ME. *Clinical Phonocardiography and External Pulse Recording*. 4th ed. Chicago: Year Book Publishers, Inc.; 1985.
9. Bethell HJN, Nixon PGF. Examination of the heart in supine and left lateral positions. *Br Heart J*. 1973;35:902–907.
10. Fowler NO, Adolph RJ. Fourth sound gallop or split first sound? *Am J Cardiol*. 1972;30:441–444.
11. Dock W. Loud presystolic sounds over the jugular veins associated with high venous pressure. *Am J Med*. 1956;20:853–859.
12. Perloff JK. Auscultatory and phonocardiographic manifestations of pulmonary hypertension. *Prog Cardiovasc Dis*. 1967;9(4):303–340.
13. Leatham A. *Auscultation of the Heart and Phonocardiography*. 2nd ed. Edinburgh: Churchill Livingstone; 1975.
14. Jordan MD, Taylor CR, Nyhuis AW, Tavel ME. Audibility of the fourth heart sound: relationship to presence of disease and examiner experience. *Arch Intern Med*. 1987;147:721–726.
15. Ozawa Y, Smith D, Craige E. Origin of the third heart sound. I. Studies in dogs. *Circulation*. 1983;67(2):393–398.
16. Ozawa Y, Smith D, Craige E. Origin of the third heart sound. II. Studies in human subjects. *Circulation*. 1983;67(2):399–404.
17. Kono T, Rosman H, Alam M, Stein PD, Sabbah HN. Hemodynamic correlates of the third heart sound during the evolution of chronic heart failure. *J Am Coll Cardiol*. 1993;21:419–423.
18. Ishimitsu T, Smith D, Berko B, Craige E. Origin of the third heart sound: comparison of ventricular wall dynamics in hyperdynamic and hypodynamic types. *J Am Coll Cardiol*. 1985;5:268–272.
19. Van de Werf F, Boel A, Geboers J, et al. Diastolic properties of the left ventricle in normal adults and in patients with third heart sounds. *Circulation*. 1984;69(6):1070–1078.
20. Tribouilloy CM, Enriquez-Sarano M, Mohty D, et al. Pathophysiologic determinants of third heart sounds: a prospective clinical and Doppler echocardiographic study. *Am J Med*. 2001;111:96–102.
21. Shah SJ, Marcus GM, Gerber IL, et al. Physiology of the third heart sound: novel insights from tissue doppler imaging. *J Am Soc Echocardiogr*. 2008;21(4):394–400.
22. Glower DD, Murrah RL, Olsen CO, Davis JW, Rankin MS. Mechanical correlates of the third heart sound. *J Am Coll Cardiol*. 1992;19:450–457.
23. Shah PM, Gramiak R, Kramer DH, Yu PN. Determinants of atrial (S4) and ventricular (S3) gallop sounds in primary myocardial disease. *N Engl J Med*. 1968;278(14):753–758.
24. Shah PM, Yu PN. Gallop rhythm: hemodynamic and clinical correlation. *Am Heart J*. 1969;78(6):823–828.
25. Homma S, Bhattacharjee D, Gopal A, Correia J. Relationship of auscultatory fourth heart sound to the quantitated left atrial filling fraction. *Clin Cardiol*. 1991;14:671–674.
26. Iga K, Konishi T. Intermittently audible the "third heart sound" as a sign of complete atrioventricular block in patients with a VVI pacemaker. *Int J Cardiol*. 1999;71:135–139.
27. Van de Werf F, Geboers J, Kesteloot H, De Geest H, Barrios L. The mechanisms of disappearance of the physiologic third heart sound with age. *Circulation*. 1986;73(5):877–884.

28. Gadsboll N, Hoilund-Carlsen PF, Nielsen GG, et al. Interobserver agreement and accuracy of bedside estimation of right and left ventricular ejection fraction in acute myocardial infarction. *Am J Cardiol*. 1989;63:1301–1307.
29. Mattleman SJ, Hakki AH, Iskandrian AS, Segal BL, Kane SA. Reliability of bedside evaluation in determining left ventricular function: correlation with left ventricular ejection fraction determined by radionuclide ventriculography. *J Am Coll Cardiol*. 1983;1(2):417–420.
30. Patel R, Bushnell DL, Sobotka PA. Implications of an audible third heart sound in evaluating cardiac function. *West J Med*. 1993;158:606–609.
31. Marcus G, Vessey J, Jordan MV, et al. Relationship between accurate auscultation of a clinical useful third heart sound and level of experience. *Arch Intern Med*. 2006;166:617–622.
32. Gadsboll N, Hoilund-Carlsen PF, Nielsen GG, et al. Symptoms and signs of heart failure in patients with myocardial infarction: reproducibility and relationship to chest X-ray, radionuclide ventriculography and right heart catheterization. *Eur Heart J*. 1989;10:1017–1028.
33. Zema MJ, Restivo B, Sos T, Sniderman KW, Kline S. Left ventricular dysfunction: bedside Valsalva manoeuvre. *Br Heart J*. 1980;44:560–569.
34. Harlan WR, Oberman A, Grim R, Rosati RA. Chronic congestive heart failure in coronary artery disease: clinical criteria. *Ann Intern Med*. 1977;86(2):133–138.
35. Marcus GM, Michaels AD, de Marco T, McCulloch CE, Chatterjee K. Usefulness of the third heart sound in predicting an elevated level of B-type natriuretic peptide. *Am J Cardiol*. 2004;93:1312–1313.
36. Narain VS, Puri A, Gilhotra HS, et al. Third heart sound revisited: a correlation with N-terminal pro brain natriuretic peptide and echocardiography to detect left ventricular dysfunction. *Indian Heart J*. 2005;57:31–34.
37. Tierney WM, Fitzgerald J, McHenry R, et al. Physicians' estimates of the probability of myocardial infarction in emergency room patients with chest pain. *Med Decis Making*. 1986;6:12–17.
38. Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. *N Engl J Med*. 1977;297:845–850.
39. Goldman L, Caldera DL, Southwick FS, et al. Cardiac risk factors and complications in non-cardiac surgery. *Medicine*. 1978;57(4):357–370.
40. Ishikawa M, Sakata K, Maki A, Mizuno H, Ishikawa K. Prognostic significance of a clearly audible fourth heart sound detected a month after an acute myocardial infarction. *Am J Cardiol*. 1997;80(5):619–621.
41. Gupta S, Michaels AD. Relationship between accurate auscultation of the fourth heart sound and level of physican experience. *Clin Cardiol*. 2009;32(2):69–75.
42. Aronow WS, Kronzon I. Correlation of prevalence and severity of valvular aortic stenosis determined by continuous-wave Doppler echocardiography with physical signs of aortic stenosis in patients aged 62 to 100 years with aortic systolic ejection murmurs. *Am J Cardiol*. 1987;60:399–401.
43. Kavalier MA, Stewart J, Tavel ME. The apical A wave versus the fourth heart sound in assessing the severity of aortic stenosis. *Circulation*. 1975;51:324–327.
44. Eagle KA, Quertermous T, Singer DE, et al. Left ventricular ejection fraction: physician estimates compared with gated blood pool scan measurements. *Arch Intern Med*. 1988;148:882–885.
45. Lee DCS, Johnson RA, Bingham JB, et al. Heart failure in outpatients: a randomized trial of digoxin versus placebo. *N Engl J Med*. 1982;306:699–705.
46. Likoff MJ, Chandler SL, Kay HR. Clinical determinants of mortality in chronic congestive heart failure secondary to idiopathic dilated or to ischemic cardiomyopathy. *Am J Cardiol*. 1987;59:634–638.
47. Folland ED, Kriegel BJ, Henderson WG, Hammermeister KE, Sethi GK. Implications of third heart sounds in patients with valvular heart disease. *N Engl J Med*. 1992;327:458–462.
48. Barlow JB. Some observations on the atrial sound. *S Afr Med J*. 1960;34:887–892.

49. Cohen LS, Mason DT, Braunwald E. Significance of an atrial gallop sound in mitral regurgitation: a clue to the diagnosis of rupture chordae tendineae. *Circulation*. 1967;35:112–118.
50. DePace NL, Nestico PF, Morganroth J. Acute severe mitral regurgitation: pathophysiology, clinical recognition, and management. *Am J Med*. 1985;78:293–306.
51. Hultgren HN, Hancock EW, Cohn KE. Auscultation in mitral and tricuspid valvular disease. *Prog Cardiovasc Dis*. 1968;10(4):298–322.